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ment depends on common signals and factors.

Asymmetric mRNA localization is crucial in establishing polarity in developing *Drosophila* eggs and early embryos. During egg (oocyte) development, mRNAs expressed in a cluster of interconnected auxiliary cells (nurse cells) are first transported into the oocyte and then targeted to specific locations within the oocyte (Fig. 1a). Once there, some of these maternal mRNAs are translated more or less immediately; their protein products help to lay down the oocyte's two 'polarity axes' (anterior–posterior and dorsal–ventral). Other localized maternal mRNAs, such as *bicoid*, are translated in the early embryo rather than the oocyte, producing a gradient of bicoid protein. This gradient helps to establish embryonic polarity by directing the initial expression of embryonic patterning genes in 'precellular blastoderm embryos' — an early stage of development in which thousands of cell nuclei are distributed around the surface of the embryo and share a common cytoplasm (Fig. 1b).

The first such patterning genes to be expressed are the gap genes, and their products form short-range gradients that subdivide the embryo into broad domains. The gap mRNAs are distributed uniformly in the cytoplasm at the surface of the embryo, and this is thought to facilitate the formation of short-range gradients extending across several nuclei[2]. The gap proteins in turn control the expression of the pair-rule genes, which further subdivide the embryo. In contrast with the unlocalized gap mRNAs, pair-rule mRNAs are targeted apically, that is, to the cytoplasm just above the nuclei that express the mRNAs[3]. This decreases the lateral diffusion of the encoded proteins, helping to refine the pattern of pair-rule gene expression and to promote the subdivision of the embryo into segments once cells have formed.

There are hints that some of the steps involved in localizing maternal mRNAs in oocytes and pair-rule mRNAs in early embryos depend on common mechanisms. First, an analysis of mRNA localization in early embryos that lack large sections of their genetic material indicated that the *trans*-acting factors required for apical localization must be maternally derived rather than expressed in the embryo[4]. Second, pair-rule mRNAs are transported to the apical cytoplasm along tracks known as microtubules, by motor proteins that move towards the 'minus' end of microtubules[5]. Similarly, the localization of several maternal mRNAs in oocytes depends on microtubules, and accumulation is directed towards the minus end[6].

Bullock and Ish-Horowicz[1] test the idea directly by investigating whether the localization machinery in blastoderm embryos can target maternal mRNAs (rather than the usual pair-rule mRNAs) to an apical location. They find that maternal messages such as *K10*, *gurken*, *bicoid*, *oskar* and *nanos* are recognized by the embryonic machinery and localized preferentially to the apical cytoplasm. Conversely, pair-rule mRNAs that are expressed artificially during oogenesis accumulate in the oocyte in a pattern resembling that of *K10* and several other maternal mRNAs.

Two other lines of evidence indicate that at least some of the same *trans*-acting factors must be used during oogenesis and embryo development. The first comes from the authors' studies[1] of two proteins, Bicaudal D and Egalitarian, which are required for mRNA localization during egg development and form a complex that accumulates at the minus ends of microtubules in oocytes[7,8]. Bullock and Ish-Horowicz[1] find that these proteins are also enriched apically in embryos and localize with a component of a minus-end-directed motor protein, dynein. Moreover, both proteins are required for the apical localization of pair-rule (naturally expressed) and maternal (artificially expressed) mRNAs in early embryos.

Second, Bullock and Ish-Horowicz[1] studied *cis*-acting localization signals. They show that the shortest RNA signal known to be active during egg development, the *K10TLS*, can also target normally unlocalized mRNAs to an apical location in embryos. This RNA signal is predicted to fold into a shape known as a 'stem–loop' structure. Mutations that disrupt this structure block the localization of *K10* during egg development; compensatory mutations that restore the structure also restore localization[9]. Bullock and Ish-Horowicz[1] find that these disrupting and compensatory mutations have the same effects on mRNA targeting in embryos, arguing that the factors that recognize the stem–loop in eggs are likely to be present in embryos, too. Conversely, pair-rule transcripts that lack the minimum apical-localization element fail to accumulate in the oocyte when expressed during oogenesis.

The simplest explanation is that the same signals and factors are used to recognize and localize different mRNAs in different developmental contexts. If true, one might expect that mRNAs that show similar localization patterns in oocytes or embryos would have similar signals. Indeed, the *orb* mRNA, which is localized similarly to *K10* in oocytes, contains a localization element that is closely related in sequence and structure to the *K10TLS*[9]. Moreover, a 150-nucleotide element involved in targeting *oskar* mRNA during oogenesis[10] has a stem–loop structure resembling that in the *K10TLS*, and the *K10TLS* can substitute for this element[9].

However, *orb* (and *oskar*) may be the exception rather than the rule: the localiza-